

Pituitary Stalk Lesions: The Mayo Clinic Experience

Adina F. Turcu, Bradley J. Erickson, Eleanor Lin, Sonsoles Guadalix, Kara Schwartz, Bernd W. Scheithauer,[†] John L. D. Atkinson, and William F. Young, Jr.

Division of Endocrinology (A.F.T., W.F.Y.), Department of Radioogy (B.J.E., K.S.), Department of Anatomic Pathology (B.W.S.), and Department of Neurologic Surgery (J.L.D.A.), Mayo Clinic Rochester, Rochester, Minnesota 55905; Division of Endocrinology (E.L.), Massachusetts General Hospital, Boston, Massachusetts 02114; and University Hospital 12 de Octubre (S.G.), 28041 Madrid, Spain

Context: Pituitary stalk lesions have various etiologies, often not clinically apparent. Pathological samples from these lesions are rarely obtained, because of the critical location and function of the hypophyseal stalk.

Objectives: The purpose of this study was to characterize the etiological spectrum of pituitary stalk lesions seen at Mayo Clinic Rochester over 20 years and to determine whether specific magnetic resonance imaging (MRI) characteristics could provide clinician guidance with regard to the etiology of infundibular lesions.

Design: A retrospective review of patients with pituitary stalk lesions seen at Mayo Clinic Rochester between 1987 and 2006 was conducted. Demographic, clinical presentation, imaging, laboratory, operative, and pathology data were reviewed and are reported using descriptive statistics.

Results: Of the 152 pituitary stalk lesions included, 49 (32%) were neoplastic, 30 (20%) were inflammatory, 13 (9%) were congenital anomalies, and 60 (39%) were of unclear etiology. Diabetes insipidus was diagnosed in 43 (28%) of the 152 patients, and 49 (32%) patients had at least one anterior pituitary hormone deficit. Secondary hypogonadism was the most common endocrine deficiency. Eleven of 13 congenital lesions were round in appearance and 5 of 7 patients with neurosarcoidosis confirmed by pathology had a uniformly thickened pituitary stalk on MRI. There were no statistically significant correlations between hypopituitarism and the pattern of enhancement or size of the lesion.

Conclusions: Findings on MRI remain key in guiding the diagnosis of pituitary stalk lesions, particularly when used in conjunction with other clinical clues. There are no good imaging predictors for hypopituitarism, making clinical evaluation of all patients with pituitary stalk lesions crucial. (*J Clin Endocrinol Metab* 98: 1812–1818, 2013)

Lesions of the pituitary stalk provide the clinician with a challenging diagnostic conundrum. As a result of advances in magnetic resonance imaging (MRI) technology, pituitary stalk lesions may be identified either incidentally or during evaluation for symptoms related to hypothalamic-pituitary dysfunction. Pathological processes that involve the hypophyseal stalk may extend from the hypothalamus and/or the pituitary gland or may be limited to the stalk itself. The etiological spectrum of these lesions is broad and can be divided in 3 categories: neoplastic, inflammatory/infectious, and congenital (1). Because of

the critical location and role of the pituitary stalk, mass lesions in this area are not often biopsied, and the diagnosis may be based on clinical evaluation and imaging clues. Herein we review the Mayo Clinic experience with pituitary stalk lesions in the era of contemporary imaging.

Materials and Methods

We conducted a retrospective review of patients with pituitary stalk lesions evaluated at Mayo Clinic Rochester between 1987

ISSN Print 0021-972X ISSN Online 1945-7197

Printed in U.S.A.

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Received December 11, 2012. Accepted February 28, 2013.

First Published Online March 26, 2013

[†] Deceased, September 20, 2011.

Abbreviations: DI, diabetes insipidus; MRI, magnetic resonance imaging.

and 2006. After institutional review board approval, we performed a text search of the radiology reports for “pituitary stalk” and “infundibulum” for all patients who had head MRI performed during the study period ($n = 150,045$). Those patients who had a magnetic resonance angiogram as part of their head MRI were excluded because “infundibulum” was a term commonly used to refer to vascular configurations other than the pituitary stalk. The 2700 head MRI radiology reports identified with this search strategy were individually reviewed in detail. Exclusion from the study was based on the following criteria: patients with inadequate imaging; patients with a normal pituitary stalk (the radiology report stated “normal pituitary stalk” or “normal infundibulum”; typical criteria used: less than 3 mm in diameter); patients with prior pituitary-directed radiation therapy or prior pituitary surgery who did not have head MRI preceding the procedure available for review. In the remaining 152 patients, the initial head MRI that identified a pituitary stalk abnormality was used for characterizing the lesion. A radiologist participant in the study individually reviewed each of these 152 head MRI scans and, without looking at the clinical context, although not formally blinded, assigned a pattern of enhancement to each stalk lesion (Figure 1).

Diagnosis assignment

We reviewed all available laboratory, pathological, clinical evaluation, and follow-up radiographic data. A diagnosis was deemed as certain in patients in whom tissue from the pituitary stalk area was obtained, either by biopsy or excision of the lesion of interest. Probable diagnoses were attributed to the following cases: (1) a tissue biopsy sample obtained from other areas of the brain and the lesion involving the pituitary stalk had a similar radiographic appearance; (2) tissue obtained from an extracranial site or, in the absence of tissue, when follow-up images, laboratory, and clinical evaluation strongly suggested a specific

diagnosis (eg, rapidly enlarging lesions in the presence of a known malignancy were suggestive of a metastatic tumor, or infiltrative lesions responsive to glucocorticoid therapy in patients with documented granulomatous disease were considered to have the same etiology); (3) ectopic pituitary tissue: the presence of a focus of increased T1 signal within the pituitary stalk, with absence of the “bright spot” in the typical location; and (4) Rathke cleft cysts: nonenhancing well-demarcated rounded lesions with homogeneously hyperintense T1 signal and hypointense T2 signal, arising from the midline between the anterior and posterior pituitary lobes. Diagnoses were labeled as “unknown” if the MRI characteristics were nonspecific and additional elements to support a diagnosis were absent or insufficient.

Hormonal evaluation

Secondary hormonal deficiencies were diagnosed based on low levels of the primary hormones with low or inappropriately normal corresponding trophic pituitary hormones. In patients with adrenal insufficiency, the cosyntropin stimulation test was often used for confirmation. GH and IGF-1 were measured in some patients, without further dynamic confirmatory tests in any of them. Diabetes insipidus (DI) was diagnosed based on typical signs and symptoms and documented with the simultaneous measurement of serum and urinary osmolality and sodium; water deprivation tests were performed in some patients.

Statistics

The paired t test was used to compare means of continuous variables. The Fisher exact test was used to compare proportions between groups. A value of $P \leq .05$ was considered statistically significant.

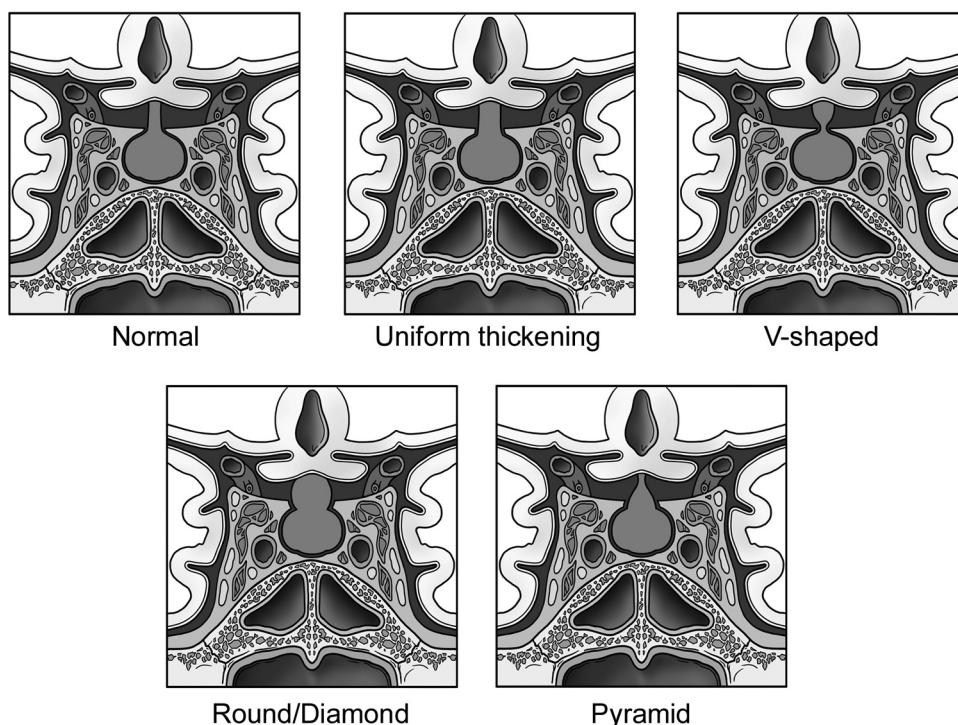


Figure 1. Imaging configurations of pituitary stalk abnormalities on MRI.

Table 1. Etiologies of Pituitary Stalk Lesions in 92 Patients

Congenital (<i>n</i> = 13)	Inflammatory (<i>n</i> = 30)	Neoplastic (<i>n</i> = 49)
Ectopic neurohypophysis (9)	Neurosarcoidosis (11)	Craniopharyngioma (5)
Normal variant (1)	Langerhans cell histiocytosis (7)	Pituitary adenoma (8)
Rathke cleft cyst (2)	Lymphocytic hypophysitis (4)	Metastatic lymphoma (9)
Pituitary cyst (1)	Erdheim Chester disease (3)	Metastatic breast cancer (6)
	Wegener hypophysitis (2)	Metastatic lung adenocarcinoma (3)
	Xanthoma disseminatum (2)	Metastatic small cell lung cancer (5)
	Lupus cerebritis (1)	Germinoma (6)
		Astrocytoma (4)
		Neuronal neoplasm (1)
		Metastasis of unknown origin (1)
		Polyclonal hypergammaglobulinemia (1)

Results

Demographics and etiological spectrum

Of the 152 patients, 91 (60%) were women. The mean age at diagnosis was 44 years (range, 2–82 years), and 17 (11%) patients were younger than 21 years. The pituitary stalk lesion was incidentally discovered in 48 (32%) patients. Tissue biopsy samples from the pituitary stalk itself or other brain lesions with similar appearance were obtained in 37 patients. Of these, one contained pituitary tissue without diagnostic abnormalities and a second one revealed glial atypia suggestive, but not diagnostic, of infiltrating glioma. An additional biopsy of an enlarging mass involving the infundibulum and hypothalamus was attempted via the foramen of Monro but was aborted because of poor visibility through the endoscope. Postoperatively, this patient developed transient hemiparesis and expressive aphasia. The diagnosis was aided by tissue obtained from peripheral lesions in 25 patients (15 with infiltrative disorders and 10 with malignant tumors) or cerebrospinal fluid samples in four patients with lymphoma.

The cause of the pituitary stalk lesion was identified in 92 (61%) patients and was attributed to neoplastic lesions in 49 (32%) patients, inflammatory lesions in 30 (20%) patients, and congenital anomalies in 13 (9%) patients (Table 1). In 60 (39%) patients, the etiology of the pituitary stalk lesion remained unknown. Of the neoplastic lesions, 13 (27%) were benign: pituitary adenoma in 8 patients and craniopharyngioma in 5 patients. Metastases accounted for most malignant lesions (25 of 36 [69%]). Of these, 15 were solid cancers and included breast carcinoma in 6 patients, small cell lung carcinoma in 5 patients, adenocarcinoma in 3 patients, and an indeterminate primary source in 1 patient. The pituitary stalk lesion was attributed to neoplasms of lymphoid tissue in 10 patients. Local primary tumors included 6 germinomas and 4 astrocytomas (Table 1). Neurosarcoidosis was the most common diagnosis in the inflammatory category, with 11 patients, followed by Langerhans cell histiocytosis, lymphocytic hypophysitis, Erdheim Chester disease, Wegener hypophysitis, xanthoma-

tous hypophysitis, and lupus hypophysitis (Table 1). Ectopic neurohypophysis dominated the congenital category, with 9 (69%) of the 13 patients (Table 1).

Clinical findings

DI was diagnosed in 43 patients (28% of the entire cohort; 51% of those specifically evaluated for DI) (Table 2). Of all patients, 49 (32%) had at least 1 anterior pituitary hormonal deficit; 32 (21%) patients had 2 or more anterior pituitary axes affected. Secondary hypogonadism was the most common endocrine deficiency, being present in 44 patients (29% of the entire cohort; 51% of those tested). Of the patients, 32 (21% of the entire cohort; 29% of those tested) had secondary hypothyroidism. Secondary adrenal insufficiency was present in 23 patients (15% of the entire cohort; 23% of tested patients) (Table 2) and was accompanied by at least 1 other hormonal dysfunction in all patients. IGF-1 and GH were measured in 74 patients and found to be low in 28 of them (38%), 9 of whom had panhypopituitarism. Three additional patients could not be evaluated, presenting to us while receiving GH replacement therapy. Of the 42 patients with DI, 30 also had anterior pituitary hormonal deficiencies. The serum prolactin concentration was elevated above the upper limit of the reference range in 29 of 95 (30.5%) patients in whom it was measured. Twenty-eight patients had no hormonal evaluation.

Visual field deficits were documented with quantitative perimetry in 16 (11%) of all patients; the mean hypophy-

Table 2. Hormonal Evaluation in Patients With Pituitary Stalk Lesions

	Thyroid Axis	Adrenal Axis	Gonadal Axis	ADH
Deficiency documented	32	23	44	43
Intact	80	79	42	42
Incomplete testing or unable to assess	10	13	18	NA
Not tested	30	37	48	67

Abbreviations: ADH, antidiuretic hormone; NA, not applicable.

seal stalk lesion diameter in these 16 patients was 6.8 mm compared with 5 mm in those without visual field deficits ($P = .02$). Visual status was formally assessed with quantitative perimetry in 54 (35.5%) patients.

MRI characteristics

The MRI patterns of lesional enhancement included uniform, V-shaped, round or diamond, and pyramidal (Figure 1). The characteristics of each etiological group are shown in Table 3. In 115 (76%) patients, the abnormality on imaging was limited to the pituitary stalk; it extended to the sella in 18 (12%) patients, involved the hypothalamus in 15 (10%) patients, and involved all 3 anatomic areas in 4 (3%) patients. The mean maximal lesional diameter was 5.2 mm (range, 2–22 mm).

The strongest association with a specific pattern of gadolinium enhancement on head MRI was that for congenital lesions; with one exception, all such lesions were round ($P < .001$). The most frequently encountered diagnosis in this category was ectopic neurohypophysis. Five of 7 patients with neurosarcoidosis confirmed by pathology presented with a uniformly thickened pituitary stalk on MRI; a similar trend (6 of 11 patients) was observed when patients with suspected neurosarcoidosis (but not confirmed with tissue biopsy) were also included. Two patients with xanthoma disseminatum involving the hypophyseal stalk were identified in our series, and both had a pyramidal pattern of enhancement on MRI. None

of the other inflammatory lesions had a characteristic pattern of enhancement on contrast MRI. No infectious lesions with pituitary stalk involvement were found in our case series. Of the neoplastic lesions, metastatic lymphomas to the pituitary stalk most frequently enhanced in a V-shaped pattern on MRI. Most metastatic solid cancers were either V-shaped or round on contrast-enhanced MRI. There was no direct correlation between a specific pattern of gadolinium enhancement on MRI and either germinomas, astrocytomas, or craniopharyngiomas (Table 3).

There was no direct relationship between the pattern of enhancement and the extent of the lesions. In addition, we were not able to identify a correlation between hypopituitarism and the pattern of enhancement or size of the lesion. In contrast, the extent of the lesions was associated with hypopituitarism as follows: while only one third of lesions limited to the stalk resulted in hypopituitarism, 50% of the lesions with sellar extension ($P = .19$), 73% of those with hypothalamic extension ($P = .004$), and 100% ($P = .014$) of the lesions affecting all 3 anatomical structures resulted in hypopituitarism.

Discussion

In this retrospective case review, we describe the etiological spectrum of pituitary stalk lesions seen in our institu-

Table 3. Patterns of Gadolinium Enhancement of Pituitary Stalk Lesions on Brain MRI

Diagnosis	No Enhancement	Pattern of Enhancement ^a					Total
		Uniform	V-Shaped	Round/Diamond	Pyramid		
Inflammatory							
Neurosarcoid	0	6	4	1	0		11
Langerhans cell histiocytosis	0	0	2	2	3		7
Lymphocytic hypophysitis	0	0	2	2	0		4
Erdheim Chester disease	0	0	2	0	1		3
Wegener hypophysitis	0	1	0	0	1		2
Xanthoma disseminatum	0	0	0	0	2		2
Lupus cerebratitis	0	1	0	0	0		1
Neoplastic							
Metastatic lymphoma	0	1	6	2	0		9
Polyclonal gammopathy	0	0	0	1	0		1
Metastatic solid carcinoma	0	1	6	6	2		15
Pituitary adenoma	0	3	1	0	4		8
Craniopharyngioma	0	1	2	2	0		5
Germinoma	0	2	1	3	0		6
Astrocytoma	1	0	2	1	0		4
Neuronal neoplasm	0	0	0	1	0		1
Congenital							
Rathke cleft cyst/pituitary cyst	0	0	0	3	0		3
Ectopic neurohypophysis	1	0	0	7	1		9
Normal variant	0	0	0	1	0		1
Total	2	16	28	32	14		92

^a Patterns of enhancement are shown in Figure 1.

tion. The literature contains very few case series on pituitary stalk lesions. Hamilton et al (2) were the first to publish a large case series with a direct focus on pituitary infundibular lesions. They included 44 adults and 21 pediatric patients. Tumor and tumor-like lesions predominated in the adult group. Neoplastic lesions were also the overall leading diagnosis in our study. However, in the previous study (2) Langerhans cell histiocytosis was considered “tumor-like,” whereas we classified it as an inflammatory disorder. It is important to recognize that the predominance of neoplastic processes in our case series does not necessarily imply a higher prevalence among pituitary stalk lesions, but rather that in such patients, especially when metastases were present, a tissue-based diagnosis was more likely to be pursued. In addition, because of the risks associated with obtaining a pituitary stalk biopsy, only a small subset of patients (24%) had their diagnoses proven by histopathology. In the series of Hamilton et al (2), 26 of 65 patients (40%) had their diagnoses established by histopathology, although it is not clear whether the tissue was obtained from the pituitary stalk lesion in all of their patients. In a series of 11 patients with pituitary stalk lesions reported by Trabelsi et al (3), 6 had no clear etiology, 3 were caused by inflammatory or infectious processes, and 2 were metastases. Congenital

lesions occurred most frequently in children in the series of Hamilton et al (2). In our study, only 11% of patients were younger than 21 years old, with no clear predominance of a specific diagnostic category in this group.

From an endocrine perspective, we found that more than one quarter of our patients had DI and nearly one third had at least one anterior pituitary hormonal deficiency. Many of our patients had no endocrinological evaluation, and it is therefore likely that our study underestimates the incidence of pituitary dysfunction in patients with infundibular lesions. Secondary adrenal insufficiency was the least common condition, and, when present, was always associated with deficiency of at least one other axis. Thyroid function was most commonly tested, whereas GH status was least frequently assessed. Hypopituitarism was more likely if the lesion extended to either the sella or the hypothalamus and occurred in all patients with involvement of both these structures. However, neither the pattern of MRI enhancement nor the size of the lesion predicted hypophyseal dysfunction. Thus, all patients with pituitary stalk lesions should have clinical and biochemical assessments for both anterior and posterior hormonal deficiencies.

Most congenital pituitary stalk lesions appeared round on MRI. The most frequently encountered diagnosis in

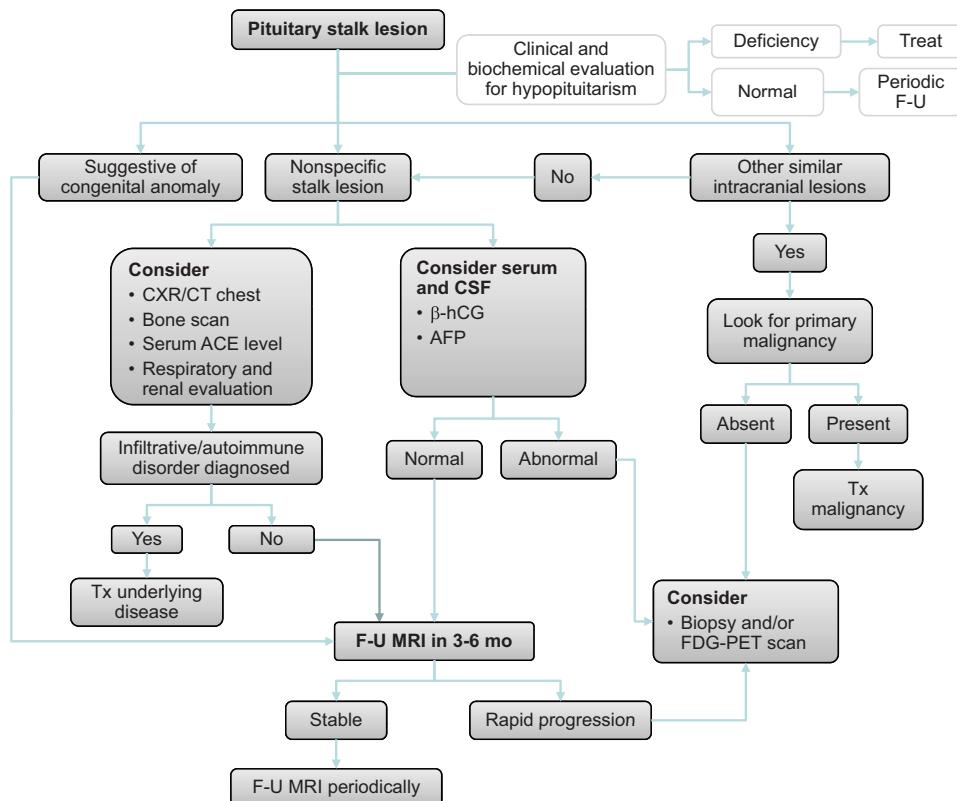


Figure 2. Suggested algorithm for diagnosis and management of pituitary stalk lesions. ACE, angiotensin-converting enzyme; AFP, α -fetoprotein; CT, computed tomography; CSF, cerebrospinal fluid; CXR, chest X-ray; FDG-PET, fludeoxyglucose positron emission tomography; F-U, follow-up; hCG, human chorionic gonadotropin; Tx, treat.

this category was ectopic neurohypophysis. The characteristic bright signal of the neurohypophysis on MRI in these patients is typically detected in the floor of the third ventricle or along the infundibulum. The sella turcica and anterior pituitary may be small and the infundibular stalk is absent or hypoplastic (4).

Most patients with neurosarcoidosis presented with a uniformly thickened pituitary stalk on MRI. Contrast-enhanced MRI is the preferred imaging test in central nervous system sarcoidosis (5). In a series of 29 patients with neurosarcoidosis, 38% showed meningeal enhancement, and 43% had multiple white-matter lesions (6). Pituitary stalk thickening and hypophyseal and hypothalamic infiltration have also been described (7).

Xanthoma disseminatum involving the hypophyseal stalk had a pyramidal pattern of enhancement on MRI. This rare histiocytosis syndrome is characterized by disseminated xanthomatous lesions that involve the skin, particularly the flexor folds and eyelids; the central nervous system, respiratory tract, and gastrointestinal tract may also be affected (8, 9). The clinical course of xanthoma disseminatum is usually benign, with spontaneous resolution of the cutaneous lesions over several years; however, involvement of important anatomical structures, such as the pituitary stalk, may cause serious complications (10, 11). For example, in our series, a 47-year-old woman presented with visual field deficits and panhypopituitarism; her head MRI showed an expansile process involving the sella turcica that was invading both cavernous sinuses, eroding the dorsum sella, and involuting into the prepontine cistern. Transsphenoidal resection followed by histopathological analysis revealed the diagnosis.

Of the neoplastic lesions, lymphomatous involvement of the pituitary stalk most frequently enhanced in a V-shaped pattern on MRI. Most metastatic solid cancers were either V-shaped or round on contrast-enhanced MRI. Although brain metastases account for more than half of intracranial tumors, metastases to the pituitary stalk have been reported rarely (12, 13). In our study, the most common primary tumors were breast and lung carcinomas, the same primary malignancies that typically metastasize to the brain (14) and to the sella (15–17). Schubiger and Haller (13) studied the MRI characteristics of 7 symptomatic hypothalamic-pituitary metastases and found that 4 also involved the suprasellar area. They were all dumbbell-shaped, with a small bridge of tissue connecting the sellar and suprasellar portions. Only 1 of these 7 tumors was located centrally in the infundibulum (13). We found no direct correlation between a specific pattern of gadolinium enhancement on MRI and either germinomas, astrocytomas, or craniopharyngiomas.

In this series of pituitary stalk lesions, the largest reported to date, we have characterized the lesions with regard to the etiological spectrum and radiological appearance. In addition to its retrospective nature, our study has several limitations. A referral center bias may have affected the types of cases included. For example, the distribution of diagnoses we are reporting may be prone to selection bias and may underestimate many undiagnosed asymptomatic lesions or lesions with lower clinical impact. However, the referral center bias might be counterbalanced by the fairly large number of lesions (almost one third) diagnosed incidentally. The main limitation of our study is the lack of tissue-confirmed diagnoses. Given the risks associated with obtaining histological samples from the pituitary stalk, even in the group presented, the diagnosis was frequently based on other clinical findings and serial imaging. Therefore, it is prudent for the clinician to follow a stepwise process to diagnose and manage patients with pituitary stalk lesions. Our general approach to these patients is outlined in Figure 2. For the most challenging lesions, an individualized approach, guided by clinical expertise, remains the best strategy.

In conclusion, pituitary stalk lesions have various etiologies that are not always clinically apparent. MRI dedicated to the hypophyseal area remains a key tool in guiding the diagnosis, especially when used in conjunction with the clinical context. Both anterior and posterior pituitary deficiencies are frequently present in patients with infundibular lesions. There are no good imaging predictors of hypopituitarism other than the extent of the lesion to the hypothalamus and pituitary gland. Thus, clinical evaluation and biochemical testing for hypopituitarism, as well as periodic follow-up, remain imperative in all patients with pituitary stalk lesions.

Acknowledgments

Address all correspondence and requests for reprints to: Dr William F. Young Jr., Division of Endocrinology, Diabetes, Metabolism, Nutrition, and Internal Medicine, Mayo Clinic Rochester, 200 First Street Southwest, Rochester, Minnesota 55905. E-mail: young.william@mayo.edu.

Funding for this study was provided by the Mayo Clinic.

Disclosure Summary: The authors have nothing to disclose.

References

- Rupp D, Molitch M. Pituitary stalk lesions. *Curr Opin Endocrinol Diabetes Obes.* 2008;15:339–345.
- Hamilton BE, Salzman KL, Osborn AG. Anatomic and pathologic spectrum of pituitary infundibulum lesions. *AJR Am J Roentgenol.* 2007;188:W223–232.

3. Trabelsi L, Mnif M, Rekik N, et al. MRI pituitary stalk abnormalities: etiology aspects in 11 patients [in French]. *Ann Endocrinol (Paris)*. 2006;67:604–612.
4. Chen S, Leger J, Garel C, Hassan M, Czernichow P. Growth hormone deficiency with ectopic neurohypophysis: anatomical variations and relationship between the visibility of the pituitary stalk asserted by magnetic resonance imaging and anterior pituitary function. *J Clin Endocrinol Metab*. 1999;84:2408–2413.
5. Sherman JL, Stern BJ. Sarcoidosis of the CNS: comparison of unenhanced and enhanced MR images. *AJR Am J Neuroradiol*. 1990;11:915–923.
6. Zajicek JP, Scolding NJ, Foster O, et al. Central nervous system sarcoidosis—diagnosis and management. *QJM*. 1999;92:103–117.
7. Bihan H, Christozova V, Dumas JL, et al. Sarcoidosis: clinical, hormonal, and magnetic resonance imaging (MRI) manifestations of hypothalamic-pituitary disease in 9 patients and review of the literature. *Medicine (Baltimore)*. 2007;86:259–268.
8. Caputo R, Veraldi S, Grimalt R, et al. The various clinical patterns of xanthoma disseminatum. Considerations on seven cases and review of the literature. *Dermatology*. 1995;190:19–24.
9. Hisanaga Y, Akaike Y, Kuroda K. Xanthoma disseminatum with large plaques confined to the back, pulmonary involvement and multiple intestinal xanthomas. *Dermatology*. 2004;208:164–166.
10. Pinto ME, Escalaya GR, Escalaya ME, Pinto JL, Chian CA. Xanthoma disseminatum: case report and literature review. *Endocr Pract*. 2010;16:1003–1006.
11. Oka M, Oniki S, Komatsu M, et al. Xanthoma disseminatum with intracranial involvement: case report and literature review. *Int J Dermatol*. 2010;49:193–199.
12. Fassett DR, Couldwell WT. Metastases to the pituitary gland. *Neurosurg Focus*. 2004;16:E8.
13. Schubiger O, Haller D. Metastases to the pituitary-hypothalamic axis. An MR study of 7 symptomatic patients. *Neuroradiology*. 1992;34:131–134.
14. Nussbaum ES, Djalilian HR, Cho KH, Hall WA. Brain metastases. Histology, multiplicity, surgery, and survival. *Cancer*. 1996;78:1781–1788.
15. Heshmati HM, Scheithauer BW, Young WFJ. Metastases to the pituitary gland. *Endocrinologist*. 2002;12:45–49.
16. Teears RJ, Silverman EM. Clinicopathologic review of 88 cases of carcinoma metastatic to the pituitary gland. *Cancer*. 1975;36:216–220.
17. Morita A, Meyer FB, Laws ER Jr. Symptomatic pituitary metastases. *J Neurosurg*. 1998;89:69–73.



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